Date: March 3, 2006

To: Jim Anderson, DEQ NWR, Portland Harbor Section Manager

From: Jennifer Peterson, DEQ NWR, Portland Harbor Section, Toxicologist

RE: Comments on Portland Harbor RI/FS: Ecological Preliminary Risk Evaluation, Dated September 9, 2005, Prepared for The Lower Willamette Group by Windward Environmental

General Comments:

1. Overall, I thought this document did a good job at presenting site-wide risk to species that feed on fish (e.g. piscivorous birds and mammals). These species are likely exposed to harbor wide prey items. However, this PRE does not evaluate receptors that would not be expected to be exposed to an ISA wide mean concentration, which include some fish (e.g. sculpin, smallmouth bass), and invertebrates. The initial assessment utilizes site wide maximums, and therefore identifies those contaminants that do not exceed the highest concentration detected. However, this evaluation is based on limited Round 1 tissue data. Adequate area coverage and historical compositing schemes not conducive with organism home range and area use limit the conclusions that can be drawn from this evaluation, leaving some uncertainty as to whether site maximums were actually included in the Round 1 tissue results. This is an uncertainty that is not mentioned in the document, but one that was clearly articulated as a part of our Round 3 approach and data gaps exercise. Much more confidence could be placed on using this information to limit COPCs if we were conducting this exercise with more comprehensive Round 3 tissue in hand for the evaluation of fish health, and other aquatic receptors of concern. This preliminary evaluation is most useful for identifying COPCs for higher trophic level organisms (e.g. birds and mammals), and can help screen out contaminants that are not likely contaminants of concern. The exception to this is for contaminants that were not measured in round 1 tissue sampling, such as PBDEs (where are we on this?). For the evaluation of fish health, this evaluation will be better informed after additional tissue samples are taken with different compositing methodology in order to evaluate more localized effects. The addition of benthic tissue (e.g. clam and lumbriculus testing), as well as the use of sediment EPCs that are calculated with the home range and likely habitat of the receptor in mind, will help reduce these uncertainties.

2. The presentation of data is definitely skewed toward looking a site-wide risk and not site-specific risk (localized areas). Unfortunately, the data presentation makes it nearly impossible to understand where more

localized risk may occur. Minimums and maximums are presented with no location numbers, and no information is provided on how many of the data points fall above a TRV range (e.g. is there one or many samples that are above a given TRV?). This gets into some real questions about spatial scale that need to be resolved. Information that could / can be presented that could help with this understanding might include:

a. Tissue: Maps of sculpin, crayfish, clam and other fish data showing TRV exceedences at each location (and for what contaminants). Much like what is presented in Figures 6-2 to 6-13 only expanded to include more chemicals and species. This should also be done for new data coming in (e.g. Round 2 benthic tissue analysis). This could also include maps where composites exceed an acceptable tissue level for upper trophic level receptors.
   Frequency information – How many of the samples exceed TRVs for the given contaminants? - all of the sculpin samples? ½ the ISA? Only in localized areas?
   Smallmouth bass samples screening on a composite-by-composite basis in the manner above similar to Figure 6-2. *Composites should represent the home range and habitat use* (currently a data gap). Again, this would be very useful after additional tissue is collected during Round 3.

b. Surface Water: Maps and data tables with each surface water sampling location, with screening conducted on a sample by basis in order to identify contaminants of interest at each location. For some contaminants this will be one of the primary lines of evidence for evaluating risk (e.g metals and PAHs).

c. Sediment – Dietary Ingestion: Fish sediment exposure point concentrations based on the home range of the receptor (again, using the correct spatial scale). Estimates of likely area use should be made and EPCs for different areas of the river should be developed. This would be used for the dietary dose analysis, and would be most important for those contaminants where we can't use the tissue residue line of evidence (e.g. PAHs and metals). This should also be done when estimating invertebrate tissue concentrations by using a BSAF, but I am hoping we will move away from this approach and actually use site-specific data collected during Round 2.

   Through this process we can develop sediment concentrations protective of dietary dose / risk for fish and map out areas that exceed those concentrations by using all the chemistry data available. Once plotted, we can see for ourselves the areas that exceed (and relate it to home range of the different species).

d. Present results for different lines of evidence so we can see how the conclusions would change.

3. This initial PRE has focused on maximum sediment / tissue concentrations and 100% site use. Explorations moving away from those assumptions are said to "be explored" in the Comprehensive Round 1 Report. If we are to move away from 100% site use for some receptors (we need to clarify which ones, etc.) we should start these discussions prior to the submittal of the report to ensure the major assumptions are agreed to prior to that document. The data available and /or necessary to modify an organism's use (other than 100%) needs to be explored.

4. For the aquatic tissue residue TRVs, several different TRV methodologies are presented here (e.g. see Table 4-1; Dyer et al method, species sensitivity distributions, and the LWG selected tissue residue values). How will we come to decisions on the TRVs to be used for aquatic life with each method having different answers?

Specific Comments:

Page 6, Section 2.3, Identification of Exposure Pathways, Aquatic Plants, Benthic Invertebrates, Fish and Amphibians: The text above states the following complete and major pathways will be evaluated quantitatively in the BERA. Transition zone water is not mentioned as a pathway for the organisms mentioned. The pathways should include direct contact / uptake from transition zone water. This also should be evaluated for some benthic fish (e.g. sculpin). This raises the question about where these other pathways will be included in the risk assessment process. It would be beneficial to have these pathways wrapped into the rest of the eco risk process (e.g. further iterations of the risk assessment).
Also, these pathway evaluations need to be consistent among documents. The DRAFT food web model submittal has several fish ventilating pore water, and in this document it is stated that those pathways are incomplete (or won't be evaluated). Those pathways should be evaluated in any risk evaluation for direct contact as well.

Page 7, Section 2.4, Receptor Groups and Exposure Pathways: The footnote here indicates that the primary line of evidence for benthic invertebrates would be the bioassay results indicating direct toxicity. However, we have multiple lines of evidence because that evaluation does not evaluate all relevant pathways. These include:

- Chemicals that would be lost during the bioassay and sediment testing, including VOCs.

- Static bioassay tests do not represent exposure occurring from fluxing / discharging groundwater. The primary line of evidence should be the comparison of transition zone water in these areas to aquatic benchmarks such as AWQCs.
- Chemicals with high Kow values would not reach equilibrium during the test durations of the bioassay test, but may accumulate and elicit effects including (e.g. PAHs and organochlorines). A primary line of evidence for these chemicals should be the tissue residue approach.

Page 8, Section 2.5, Footnote 4: The footnote states that only sediment data from Round 1 sampling was included to select COIs. The list of additional COIs identified in Round 2 sediment is presented in table E-1 in Appendix E. This should be mentioned in other parts of the report as well. If someone misses this footnote they would be unclear that Round 1 and Round 2 data was limited to those detected in Round 1. Also, it is unclear what the historical data shows relative to the additional COIs.

How were sums calculated relative to PCBs? For example, if certain Aroclors were not detected in Round 1 sampling, but were in Round 2, were they included in the PCB sums for sediment (e.g. in calculating dietary exposure to fish, birds and mammals)? Table 2-2 shows the COIs identified for each receptor group based on this analysis. However, the total PCB values are footnoted indicating that limited Aroclors were detected in Round 1 sampling. Does this mean that Aroclors contributing to total PCB concentrations (from Round 2) in sediment were not used in this analysis (e.g. 1221 and 1268)?

Page 10, Section 3.1.1 and 3.1.2, Surface Sediment and Tissue Data: For understanding localized risk, the summary of the chemistry and tissue results (Appendix D) should include location numbers (at least where the min and max were detected).

Page 10, Section 3.1.2, Footnote 5: Locations of fish collections should be presented, and for all fish species (e.g. carp). Summary maps can easily be created from the characterization reports.

Page 11, Section 3.1.3, Surface Water Data: PCB concentrations were not available to estimate aquatic invertebrate tissue concentrations for use in the dietary exposure estimates. Since the XAD analysis is a must for estimating tissue, this is a limitation in dietary estimates. They likely underestimate PCB concentrations, as the peristaltic pumps did not achieve the necessary detection limits.

Page 14, Section 3.2.2, Last Paragraph, Summation Rules: This document is using the “Guidelines for Data Reporting”, Kennedy/Jenks et. At 2004, but it also is applying “ecological risk assessment summation rules” *that were not presented in the PRE Approach TM*. The additional summation rules include

summing total PCBs, DDTs, PAHs, chlordanes and endosulfans. It is stated that for each sampling location where "some analytes contributing to the sum are detected and some are not detected, only detected concentrations will be summed to represent the total concentration." Was there a consideration of appropriate detection limits?
It is unclear how the summation rules were carried forward to the risk assessment. Summation rules should not be used where one of the individual isomers is more toxic than the others in the sum. This could over or underestimate the risk depending on the composition. For example, alpha endosulfan has been shown to be about three times as toxic as the beta isomer of endosufan.
How are the TRVs being applied for summed chemicals? We could sum in this manner if there is an agreement to use the TRV of the most toxic isomer in the risk assessment. However, beyond the risk assessment it would be important to know the composition of the environmental sample.
Applicability of summation varies with the receptor and pathway as well as the toxicity of the individuals in the sum. The text is not specific, does this applies to water, sediment or tissue, or all media?
The text states, "total concentrations were calculated for Round 1 and Round 2 and relevant non-LWG data collected data sets per sampling location". What non-LWG datasets were included and which were not? What criteria were used? In addition to the SCRA database, do we need to review an "eco risk SCRA" database? Is this available?

Page 14, Section 3.3.3, Reduction Rules for Existing Data: Why are we using different rules for different data sets? It seems like if we want to combine with the historical data the LWG data shouldn't be reduced (or vice versa). All sample results should be reported for both LWG and non-LWG data for field duplicates – was this not done?

Page 15, Section 4.0, Effects Characterization, COIs with no TRVs: TRVs were not developed aluminum and manganese. They proposed to look at available upstream and /or state region-wide background concentration. This analysis should be presented here, as we have some of the data now. If they are elevated, we need to develop a plan for assessment (e.g. the development of TRVs).

Page 16, Section 4.1.1, Aquatic Tissue Residue TRVs, Tier 1: It appears that the TRV was selected from the 5th percentile of LOEC data only from fish, clams and crayfish and not ALL aquatic species LOEC data. It is important that other invertebrate data is included in the distribution. The text here is a little unclear, but Table 4-1 footnote B indicates only fish, clam and crayfish data were used. I believe this was not the original intention of EPA's comment.

Page 17, Section 4.1.2, Fish Dietary TRVs and Tables 4-3 and 4-4: No extrapolation factors were applied to develop NOECs from LOECs. This is

necessary for the use of the dietary approach. Specific protocol should be followed in the development of the dietary TRVs. Previous comments from EPA have emphasized the need to develop dose information for fish, not rely on concentration based TRVs. The concentration only comparison should be omitted, and toxicity data from various toxicity endpoints should be converted to an exposure dose in mg/kg/day. This information can be compiled, and fit to a cumulative distribution function similar to what was done for the aquatic TRV development. Menzie-Cura and the US Army Corps of Engineers did this analysis for PAHs. A database of NOAELs and LOAELs was compiled consisting of PAH toxicity data. These included PAH toxicity data from 15 studies on 8 species, and included various life stages (larvae, fry, juvenile, adult), various compounds (BaP, DMBA, fluor, anthr, phen), various routes of exposure (water, diet, injection), various exposure durations (single injections, weeks, months), and various toxicity endpoints (heptatic lesions, growth, immunological, reproductive). Water concentrations were converted to an exposure metric (mg/kg/d) using the Arnot and Gobas equations (2004). The data was then fit to a cumulative distribution function, and the geometric mean of the NOAEL and LOAEL was calculated for each study to estimate NOAELs where they were not reported. The data were fit to a log-logistic cumulative distribution function, which was then used to estimate protective doses below which adverse effects in most fish are unlikely. This analysis developed benchmark doses of about 0.01 to 0.2 mg/kg/day to represent doses below which toxic effects are not expected for most fish. This range is below the selected PAH LOAEL TRV reported in Table 4-3 of 1.9 (mg/kg/d). The goal should be to develop dietary NOAELs for fish that are protective of all endpoints. The methodology outlined above seems like a good model to work from. This model can be carried forward to the other chemicals for which the dietary approach was used (e.g. Table 4-3 and 4-4).

For tables 4-3 and 4-4 the final TRV selection was not clear. The selected concentration based TRVs differ from the dose TRVs. The concentration values should be converted to a dose and used in the TRV development.

Page 18, Section 4.2, Summary of TRV Analysis for Wildlife: Tissue residue concentrations in egg are presented here as a secondary line of evidence for birds. It should say that this pathway is one of the primary lines of evidence for certain contaminants, such as dioxin and dioxin-like chemicals, PCBs and DDE.

Page 18, Section 4.2.1, Wildlife Dietary TRVs: It is stated, “no extrapolation was done to derive LOAEL TRVs”. If no LOAEL is available, are we using the NOAEL value as the final TRV? Why aren’t we using extrapolation to derive LOAELs from NOAELs?

Page 20, Section 5.0, Exposure Characterization: The text here should say that exposure was not estimated for all the chemicals detected in Round 2 sediment, as listed in table E-1

Page 21, Section 1.1.1.1, Site-specific relationship between sediment and tissue chemical concentrations: The government team is currently reviewing the BSAF analysis by pulling the appropriate data and reviewing the analysis. There is not enough information presented here for us to replicate the analysis. Also, given the updated Round 2 sediment information, additional samples should be pulled in if they help resolve sculpin / area use issue. The analysis presented here focuses on the Round 1 co-located samples at the exclusion of new information in the area.

Page 22, Section 5.1.1.2, Literature Based BSAFs: I did not review the applicability of the BSAFs selected for this exercise. I am anticipating that the Round 2 analysis of sediment and invertebrate tissue will result in more relevant BSAFs for this purpose.

Page 23, Section 5.1.2, Bioaccumulation and Bioconcentration Factors: Literature derived BCFs should be reviewed. However, collection of site-specific information that would allow us to develop BCFs more relevant for this site is a data gap we should be filling during Round 3.

Page 24, Section 5.1.2, BCF Retrieval: Why were BCFs only retrieved for Aroclors 1260, 1254, 1242, and 1016? Water BCFs should be based on the individual congeners, and not on sums.

Page 25, Section 5.2.1.1, Tissue Approach: The text states “in the risk characterization 95% UCLs of chemical concentrations in tissue were used to represent less conservative EPCs”. Evaluations of tissue residues for the purposes of evaluating fish health should not include a mean value calculated from composite means, especially of the composites were taken over an area larger than a localized population. Each composite should be screened individually against a TRV, and the areas exceeding reported. Again, this is a spatial scale issue that needs to be resolved before the next iteration of the risk assessment. Development of 95% UCLs on the mean of composite samples may be more appropriate when evaluating the risk to those species feeding on the fish over large areas (e.g. human health, eagle, osprey, mink).

Page 26, Equation 5-2, Estimating Benthic Invertebrate Tissue Conc.: It is unclear why the mean OC-normalized surface sediment concentration was used to represent surface sediment concentration. The range should be represented – e.g. there may be significant area above the mean value used here, and this would underestimate benthic tissue concentration in some areas.

Page 26, Equation 5-3, Estimating Invertebrate Tissue Concentrations: The mean value shouldn’t be used for this – much of the water column concentrations are also means (temporally and spatially integrated) for a given sample area.

For water evaluations, we should be using the maximum or representing the range.

Page 30, Section 5.2.1.3, Dietary Assumptions, Fish: The average BW is used to estimate FIRs for each fish. The range of body weights should be reported; implications for the potential range of fish body weights on risk estimates should be discussed.
Page 32: Can't we estimate a body weight for sturgeon and get some risk estimates?

Pages 30-36, Section 5.2.1.3, Dietary Tissue Assumptions: Taking the maximum of potential prey items is conservative and will likely show risk. However, we should be working toward something more realistic so that we can be in agreement before the next iteration. For example, I think we can agree smallmouth bass are not feeding on brown bullhead, northern pikeminnow, carp or black crappie (at least not the size classes we sampled). So what does this analysis tell us about dietary risk to the smallmouth bass? It would seem relevant to have sculpin and crayfish represent the diet – with one dietary scenario feeding primarily on crayfish and the other on sculpin. How would that change the risk estimates?

Page 36, Section 5.2.2, Benthic Invertebrate Exposure Assessment: There is a statement here that the bioassay testing (direct toxicity testing) will be the primary line of evidence for evaluating risks to the benthic community, that the other "qualitative" lines of evidence may include "assessment of risks via the transition zone and surface water exposure pathways." These are other *pathways*, not different lines of evidence for one exposure pathway, and should be evaluated as such.

Page 37, Section 5.2.2, Benthic Exposure Assessment: 95% UCLs (on the mean) are appropriate when a mobile receptor is exposed to a media or prey. As it is feeding it will be exposed to an average concentration (represented by a 95% UCL). However, to assess risk to a species of concern, tissue residue values should not be compared to a mean of tissue residue concentrations, because effects are occurring on the population for those above the mean. Each sample composite should be compared individually to a TRV, and not averaged over the site.

Page 38, Section 5.3.1.2, Dietary Exposure Doses: I was not able to replicate the dietary doses presented. The sediment concentrations used in these equations was not presented (except in Appendix D?).

Pages 39-47, Section 5.3.2, Dietary Assumptions: Average percent moisture values for Round 1 tissue were used for all receptors to estimate wet weight food concentration in order to calculate dose. This included all tissue from round 1 including invertebrates and fish. Percent moisture should be calculated for

different species, and carried through the dietary dose equations. This is a sensitive parameter in this conversion, since it will vary the exposure concentration. How was % moisture calculated from Round 1 tissue? Was it calculated from % solids?

For most receptors, average exposure values were used to calculate risk estimates (e.g. body weight, ingestion rates). Male and female differences could be important in determining sensitivity. A range of risk estimates based on male and female body weights and ingestion rates could be presented in the baseline report or the most sensitive combination used – simply using the female body weights and ingestion rates have been used in other risk assessments.

Page 46, Section 5.3.2.6, River Otter: The carnivorous mammal prey that should be used here would be a Portland Harbor specific fish % moisture value from round 1.

Page 49, Section 6.0, Risk Characterization: The text states "the NOAEL-based HQs less than a value of 1 are assumed to represent acceptable levels of exposure and risk for the receptor / pathway / COPC combinations represented by the analysis." This statement should be limited to bird and mammal receptors. Uncertainties associated with lack of site-specific tissue (e.g invertebrates), area coverage and compositing methodologies limit the screening we can conduct on benthic invertebrate and fish receptors.

Page 51, Section 6.1.2, White Sturgeon: Why isn't an option of "dietary approach as represented by a dose" shown here?

Page 60, Section 6.3.1.1: 95% UCLs for fish tissue would be appropriate if developing an exposure point concentration (prey) for higher trophic level organisms (e.g. human health, osprey, eagle, mink) feeding on fish. However, calculating 95% UCL on a mean of composite (average) data would not be protective of fish populations from localized areas. Each individual composite sample (which actually represents a mean or average) should be compared to a TRV directly.

Page 61, Section 6.3.2.2, Use of 95% UCLs of sediment conc: It is unlikely that some fish species (e.g. sculpin, bass) would not be exposed to a mean of the entire 9 miles of ISA. Therefore, meaningful exposure areas should be used if determining 95% UCLs of a sediment area. For smallmouth bass, this area may only be ¼ of a mile. Also, sediment exposure areas may not include channel areas, which may not be likely habitat for some species.

Page 63, Section 6.3.3.2, Use of 95% UCLs of sed conc, Wildlife: See above comment on fish. It is unlikely that for some receptors (e.g. sandpiper) that they would be exposed to a mean concentration over the entire 9 miles of ISA. Smaller exposure areas are needed.

Page 64, Section 7.0, Identification of Uncertainties: Why are the HQs based on the egg-modeling pathway for birds stated as "highly uncertain"? It appears overly conservative BMFs were used to estimate egg concentrations. Further refinement of this approach should lead to more realistic results.

Page 65, Section 8.0, Conclusions: How were the many chemicals that didn't have a calculated NOAEL (some had LOAELs), fit into the statement that "COIs for which screening-level exposure estimates did not exceed NOAEL-based TRVs are not likely to represent unacceptable risk for the pathways evaluated"… handled? Were they all "screened in"?

Table 2-1, PH Work Plan Assessment Endpoint Table: Do we need to make changes to this table as a part of the Round 3 approach and data gaps planning exercise? I had some questions:
Benthic community: The tissue residue line of evidence is missing.
Juvenile Chinook and sculpin: There is some language here about biomarkers (and how they will not be collected or used as a part of the RI/FS) – is this accurate? Our original table had language accepting the use of biomarkers as an additional line of evidence.
Other species: We are identifying more data gaps related to additional tissue collection, etc.

Table 4-2, Water Quality Criteria TRVs: Why weren't the state of Oregon water quality criteria used (those currently in effect; not yet approved by EPA)?

Table 4-4- Dietary Fish TRVs: NOECs must be estimated from LOAECs for the TRV development.

Table 4-6, Wildlife Dietary TRVs and Table 4-7, Mammal Dietary TRVs: To my knowledge, these still need to be reviewed by EPA. It is unclear if the TRV values were reviewed by Parametrix to ensure they were appropriately protective, and that the literature review included all relevant studies.

Table 5-7, Estimated Invert Tissue Residues: Each surface water location should have been done separately, as some fish would not be exposed to the entire ISA. A range of modeled water-column invert tissue concentration should be presented and used in the risk estimates. Alternatively, for the PRE, the maximum water concentration could have been used.

Table 5-9, Fish Dietary EPCs Based on Sediment Conc: It is important to agree on an appropriate way to move forward from the maximum sediment concentration to more of an area-weighted average. Consensus on an appropriate area of exposure (e.g. home range) for each receptor before the next submittal is necessary. The 95% UCL on the mean will not be appropriate for all

receptors, as it would not be likely that they would be exposed to the 9 plus miles of ISA.

Table 5-14, Bird and mammal dietary prey assumptions: Prey % moisture characteristics should be specific to the prey of each receptor. For example, for osprey the % moisture should be the average of the species listed, and shouldn't include clams.

Table 5-15, Bird Dietary Exposure Doses: Several food ingestion rates are calculated using the literature to define the % moisture of the diets, and others used the Round 1 tissue average concentration. For determining a site exposure dose we are converting a dw food ingestion rate and converting it to a wet weight and should assume they are feeding from the Willamette, and use appropriate % moisture data from prey items identified in the Round 1 tissue collection. For the development of TRVs we should be using the % moisture from the study the TRV was developed.

Page 106, Table 7-1, Risk Characterization Uncertainties: How is over or underestimate and "risk driver" being defined here? Is this site-wide, or localized risk?

Appendix A, Approach for the Preliminary Risk Evaluation For Ecological Receptors: A revised approach is here (from our March 2005 comments?). Some sections have been added since the approach document (e.g. ecological summation rules). Is this supposed to be reviewed to see how our comments were interpreted, or are we concentrating on the final document?

Appendix B, Toxicity Reference Value Selection: I did not review this appendix. We should confirm that all the TRVs have been reviewed – esp. the wildlife dietary TRVs. Some selections do not make sense when the lowest NOAEL / LOAEL combination was not selected (e.g. Figure 32 for nickel; Figure 47 for chromium; Figure 49 lead; Figure 67 for butyl benzyl phthalate).

Appendix B, Fish Diet TRVs: Why aren't the diet based PAH TRVs presented here?

Appendix B, TRVs: Why were NOEC values selected that are higher than a relevant LOEC (e.g. see Figure 17 for copper)? Why weren't LOECs selected in some cases (e.g. copper)?

Appendix C, Statistical Analysis of Site-Specific Relationship Between Tissue and Sediment Chemical Concentrations, General Comment: There is not enough information presented here to replicate their analysis. Efforts have been made to obtain the data in the form necessary to review the BSAF analysis conducted here. However, this review has not occurred yet (unless Parametrix did it?).

Appendix C, Page 2-3: Relationships between sediment and tissue should be tested for significance at an alpha level of 0.1. This was commented on previous versions of the PRE approach. It appears the final PRE did not follow previous comments made, or the PRE presented in Appendix A, which does show that an alpha of 0.1 will be used. Only the ones that had a relationship at a significance of 0.05 were pursued further, which could be missing significant relationships and other contaminants. The nature of the correlation between tissue and sediment concentrations should be investigated using scatterplots and regression analysis for those that had a significance of 0.1. Also, for the regression relationships, it should not be concluded, "if the slope (m) is not significantly different from zero (with an alpha of 0.5) then the linear relationship between log-tissue and log-sediment is not significant and log-tissue can be most efficiently approximated using the geometric mean of tissue concentrations rather than modeled as a function of sediment concentrations".

Appendix C, Section 3.3, DDE in Sculpin Tissue: For BSAF analysis, non-detect values should not be treated as ½ the detection limit. The data gap here should be filled with more tissue sampling. In this case, 11 sediment and 5 tissue samples were non-detect (we need to know if these were elevated detection limits), and only 6 samples actually had co-located detections of sediment and tissue. The non-detects in this case is likely to skew the development of the relationship.

Appendix E, Evaluation of Uncertainties, Section 3.1.5, Performance of Additional Risk Analysis: The analysis of human health fish for ecological risk showed higher HQs, which may be important for some that showed up based one line of evidence (e.g. zinc, chromium and cadmium). For example, zinc was just over an HQ of one for juvenile Chinook, and close for all other species 0.6 to 0.93. Chromium shows up here, and in the 5^{th} percentile analysis (LOEC), which as one order of magnitude lower than the Appendix B NOEC and LOEC.

Appendix E, Section 3.2, Dietary Approach: Given the HQ values for TBT in this report, TBT should be analyzed for in future fish tissue sampling. This is a data gap in that right now we only have the dietary approach as one line of evidence.

Appendix E, Section 3.2.3, Availability of tox data for dietary TRVs: We should be calculating a NOAC from a LOEC if it is available. Dietary LOECs may have underestimated dietary risk and this should be revised.

Appendix E, Section 3.2.4, Maximum sed / tissue conc: The maximum concentration risk may be indicative of localized risk, which may be relevant for some receptors. This should be discussed as to where and how big these areas are likely to be within the ISA.

Appendix E, Section 4.1, Dietary Approach: The use of 100% site use for receptors in the risk assessment is listed as an uncertainty. For any receptor where 100% site use is not used, the data available to change the site use will need to be reviewed. We should be working on these issues before the next submittal.

Appendix E, Section 4.1.2, Availability of Tox Data for dietary TRVs: Where TRVs are not available, the use of a surrogate should be explored.

Appendix E, Section 4.1.3: The use of 95% UCL sediment concentrations is cited as potentially being more realistic. Were these calculated using relevant organism foraging areas (e.g. near shore versus channel) or were all sediment samples used to develop that mean?

Appendix E, Table E-4: What does the “a” footnote mean? Is it based on detection limit?

Appendix E, Table E-5: How is “risk driver” defined here? How were determinations of “underestimate” and “overestimate” made?